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Physiology

1. There are several physiological mechanisms that regulate blood pressure in the long-term, the first of which is the renin-angiotensin-aldosterone system (RAAS).

Renin-Angiotensin-Aldosterone System (RAAS)

Renin is a peptide hormone released by the granular cells of the juxtaglomerular apparatus in the kidney. It is released in response to:

- a. Sympathetic stimulation
- b. Reduced sodium-chloride delivery to the distal convoluted tubule
- c. Decreased blood flow to the kidney

Renin facilitates the conversion of angiotensinogen to angiotensin I which is then converted to angiotensin II using angiotensin-converting enzyme (ACE).

Angiotensin II is a potent vasoconstrictor. It acts directly on the kidney to increase sodium reabsorption in the proximal convoluted tubule. Sodium is reabsorbed via the sodium-hydrogen exchanger. Angiotensin II also promotes release of aldosterone.

Aldosterone promotes salt and water retention by acting at the distal convoluted tubule to increase expression of epithelial sodium channels. Furthermore, aldosterone increases the activity of the basolateral sodium-potassium ATP-ase, thus increasing the electrochemical gradient for movement of sodium ions.

More sodium collects in the kidney tissue and water then follows by osmosis. This results in decreased water excretion and therefore increased blood volume and thus blood pressure.

Anti-Diuretic Hormone (ADH)

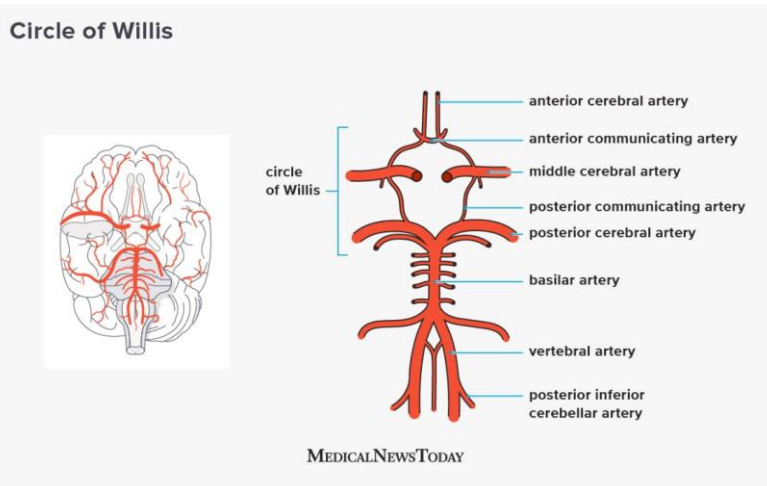
The second mechanism by which blood pressure is regulated is release of Anti Diuretic Hormone (ADH) from the OVLT of the hypothalamus in response to thirst or an increased plasma osmolarity.

ADH acts to increase the permeability of the collecting duct to water by inserting aquaporin channels (AQP2) into the apical membrane.

It also stimulates sodium reabsorption from the thick ascending limb of the loop of Henle. This increases water reabsorption thus increasing plasma volume and decreasing osmolarity.

Other factors that can affect long-term regulation of blood pressure are natriuretic peptides. These include:

- a. Atrial natriuretic peptide (ANP) is synthesised and stored in cardiac myocytes. It is released when the atria are stretched, indicating of high blood pressure.
ANP acts to promote sodium excretion. It dilates the afferent arteriole of the glomerulus, increasing blood flow (GFR). Moreover, ANP inhibits sodium reabsorption along the nephron. Conversely, ANP secretion is low when blood pressure is low.
 - b. Prostaglandins act as local vasodilators to increase GFR and reduce sodium reabsorption. They also act to prevent excessive vasoconstriction triggered by the sympathetic nervous and renin-angiotensin-aldosterone systems.
2. a . Pulmonary circulation is the system of transportation that shunts de-oxygenated blood from the heart to the lungs to be re-saturated with oxygen before being dispersed into systemic circulation. Deoxygenated blood from the lower half of the body enters the heart from the inferior vena cava while deoxygenated blood from the upper body is delivered to the heart via the superior vena cava. Both the superior vena cava and inferior vena cava empty blood into the right atrium. Blood flows through the tricuspid valve into the right ventricle. It then flows through the pulmonic valve into the pulmonary artery before being delivered to the lungs. While in the lungs, blood diverges into the numerous pulmonary capillaries where it releases carbon dioxide and is replenished with oxygen. Once fully saturated with oxygen, the blood is transported via the pulmonary vein into the left atrium which pumps blood through the mitral valve and into the left ventricle. With a powerful contraction, the left ventricle expels oxygen-rich blood through the aortic valve and into the aorta: This is the beginning of systemic circulation
- c. Two arteries, called the carotid arteries, supply blood to the brain. They run along either side of the neck and lead directly to the circle of Willis. Each carotid artery branches into an internal and external carotid artery. The internal carotid artery then branches into the cerebral arteries. This structure allows all of the blood from the two internal carotid arteries to pass through the circle of Willis.
The circle of Willis is critical, as it is the meeting point of many important arteries supplying blood to the brain. The internal carotid arteries branch off from here into smaller arteries, which deliver much of the brain's blood supply.



- d. The splanchnic circulation powerfully influences systemic arterial pressure via two distinct mechanisms. Widespread contraction of arteries in the splanchnic bed reduces blood flow to the region. The low oxygen consumption of splanchnic organs allows for a very large reduction in blood flow without producing ischemia. Arterial constriction causes dramatic increases in systemic arterial pressure and total peripheral resistance. Cardiac output also may increase due to passive discharge of stored blood from downstream veins into the central circulation. Active constriction of veins in the splanchnic organs reduces regional blood volume. This has relatively little effect on total peripheral resistance but raises cardiac output and arterial pressure by increasing central blood volume and thus cardiac preload. Generalized arterial and venous constriction in the splanchnic circulation occur mainly in response to extrinsic neural and hormonal inputs. Catecholamines, angiotensin II and vasopressin are among the most powerful hormonal vasoconstrictors. They serve this function particularly in response to major challenges to overall circulatory homeostasis, for example acute hypovolemia caused by hemorrhage.
- e. Coronary circulation is the circulation of blood in the blood vessels that supply the heart muscle (myocardium). Coronary arteries supply oxygenated blood to the heart muscle, and cardiac veins drain away the blood once it has been deoxygenated. Because the rest of the body, and most especially the brain, needs a steady supply of oxygenated blood that is free of all but the slightest interruptions, the heart is required to function continuously. Therefore its circulation is of major importance not only to its own tissues but to the entire body and even the level of consciousness of the brain from moment to moment. Interruptions of coronary circulation quickly cause heart attacks (myocardial infarctions), in which the heart muscle is damaged by oxygen starvation. Such interruptions are usually caused by ischemic heart disease (coronary artery disease) and sometimes by embolism from other causes like obstruction in blood flow through vessels.
- f. The cutaneous circulation is the circulation and blood supply of the skin. The skin is not a very metabolically active tissue and has relatively small energy requirements,

so its blood supply is different to that of other tissues. Some of the circulating blood volume in the skin will flow through will flow through arteriovenous anastomoses (AVAs) instead of capillaries. AVAs serve a role in temperature regulation. In this article we shall consider the different adaptations of the cutaneous circulation, and its role in body temperature control.

Arteriovenous Anastomoses

AVAs are low-resistance connections between the small arteries and small veins that supply and drain the skin. These allow the shunt of blood directly into the venous plexus of the skin, without it passing through capillaries. Since AVAs contain no capillary section, they are not involved in transport of nutrients to/from the tissues, but instead play a major role in temperature regulation.

3. Cardiovascular adjustment during exercise

One of the major adjustments made by the CVS during exercise is increased cardiac output ie increased pumping capacity of heart enhancing delivery of oxygen and fuel to working muscles

- Chemo/Baroreceptors detect stimuli
- The stimuli passes through the cardiovascular control centre in the medulla oblongata and stimulates it.
- There is an increased input from sympathetic nerves and withdrawal of parasympathetic nerves
- This leads to an increase in impulse at the SA node
- An increase in impulse at SA node will lead to an increase in heart rate /stroke volume which will lead to an increase in cardiac output.